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REVIEW ARTICLE

Meta-analysis of social cognition in attention-deficit/hyperactivity disorder (ADHD): comparison with healthy controls and autistic spectrum disorder

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Background. Impairment in social cognition is an established finding in autism spectrum disorders (ASD). Emerging evidence suggests that attention-deficit/hyperactivity disorder (ADHD) might be also associated with deficits in theory of mind (ToM) and emotion recognition. However, there are inconsistent findings, and it has been debatable whether such deficits persist beyond childhood and how similar social cognitive deficits are in ADHD *v.* ASD.

Method. We conducted a meta-analysis of social cognition, including emotion recognition and ToM, studies in ADHD compared with healthy controls and ASD. The current meta-analysis involved 44 studies comparing ADHD ($n = 1999$) with healthy controls ($n = 1725$) and 17 studies comparing ADHD ($n = 772$) with ASD ($n = 710$).

Results. Facial and vocal emotion recognition ($d = 0.40–0.44$) and ToM ($d = 0.43$) abilities were significantly impaired in ADHD. The most robust facial emotion recognition deficits were evident in anger and fear. Social cognitive deficits were either very subtle (emotion recognition) or non-significant (ToM) in adults with ADHD. Deficits in social cognition, especially ToM, were significantly more pronounced in ASD compared with ADHD. General cognitive impairment has contributed to social cognitive deficits in ADHD.

Conclusions. Performance of individuals with ADHD on social cognition lies intermediate between ASD and healthy controls. However, developmental trajectories of social cognition probably differ between ADHD and ASD as social cognitive deficits in ADHD might be improving with age in most individuals. There is a need for studies investigating a potential subtype of ADHD with persistent social cognitive deficits and exploring longitudinal changes in social cognition during development.

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Key words: Attention-deficit/hyperactivity disorder, autism, emotion recognition, social cognition, theory of mind.

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a heterogeneous developmental disorder characterized by inattention, impulsivity and hyperactivity. ADHD is one of the most common neurodevelopmental disorders as defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM) with over 5% of the population meeting DSM-IV criteria for this condition. While many children with ADHD present with both hyperactivity/impulsivity and inattention, subgroups (specifiers in DSM-5) presenting with pure inattention or pure hyperactivity/impulsivity have been introduced (American Psychiatric Association, 2013). The severity of symptoms and long-term course of ADHD are variable (Karam *et al.* 2015). ADHD symptoms, especially hyperactivity and impulsivity, improve with age, but some symptoms including inattentiveness, disorganization and problems in emotion regulation persist into adulthood in many individuals. Evidence also suggests that individuals with ADHD continue to present with cognitive difficulties (Alderson *et al.* 2013; Mowinckel *et al.* 2015). While some individuals with ADHD function relatively well and perceive ADHD as a condition with some positive aspects, such as increased creativity, novel problem-solving abilities and multi-tasking at will (Fleischmann & Fleischmann, 2012), the symptoms of ADHD have an overall negative impact on quality of life in many individuals (Danckaerts *et al.* 2010). This is particularly so in the context of increasing demands of modern life including competitive education and the work environment.

In addition to problems in impulse control, attention and other neurocognitive problems such as working memory, ADHD presents with emotion dysregulation

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and significant interpersonal problems including conflict with parents, siblings, peers, teachers, which can lead to rejection (Barkley, 1997; Nixon, 2001; Shaw *et al.* 2014). Indeed, social dysfunction is one of the most debilitating aspects of ADHD (Nijmeijer *et al.* 2008). Evidence also suggests that there is significant clinical overlap between ADHD and autism spectrum disorder (ASD) in terms of impairment in social functioning. It seems that there is a subgroup of patients with ADHD with very significant deficits in social functioning comparable with high-functioning ASD. For example, Greene *et al.* (1996) reported that 22% of individuals with ADHD have substantial deficits in social functioning. While there are multiple possible clinical and psychosocial determinants for these problems, the ability to recognize another person's emotions and other social cognitive abilities, which have been traditionally associated with diagnosis of ASD (American Psychiatric Association, 2013), can contribute to these difficulties. Social cognition has multiple domains including emotion recognition, theory of mind (ToM), social knowledge, attributional style and social perception (Green *et al.* 2008) and ADHD might be associated with deficits in each of these domains (Grenell *et al.* 1987; Collett & Gimpel, 2004; Uekermann *et al.* 2010; Collin *et al.* 2013; McQuade *et al.* 2014). Two domains of social cognition, recognition of emotional expressions (mostly measured based on sets of still photographs/drawings or recorded vocal expressions of six basic emotions) and ToM, the ability to attribute mental states (feelings, beliefs, intentions and desires) to others and understand and predict others' behaviour based on their mental states, have been investigated in a sizeable number of studies. However, it is not clear whether emotion recognition and ToM deficits in ADHD are comparable in severity to ASD (Buitelaar *et al.* 1999; Nydén *et al.* 2010; Baribeau *et al.* 2015). Investigating the developmental course of social cognition in ADHD is also important as there is a significant development in social cognitive abilities during adolescence and adulthood (Choudhury *et al.* 2006; Brizio *et al.* 2015). Indeed, there is conflicting evidence regarding the persistence of social cognitive deficits in ADHD beyond childhood (Miller *et al.* 2011; Bagcioglu *et al.* 2014; Ibáñez *et al.* 2014). It is also important to investigate whether social cognitive impairments in ADHD are an independent abnormality or secondary to intellectual abnormalities, as neurocognitive abilities contribute to performance of patients with neuropsychiatric disorders on social cognition tasks (Aboulafia-Brakha *et al.* 2011). Co-morbid ASD in ADHD is common and might potentially contribute to social cognitive impairment (Taylor *et al.* 2013). Conduct disorder is another important co-morbidity in ADHD (Pliszka, 1998). Conduct disorder is associated with significant interpersonal problems and might be relevant for deficits in social cognition in ADHD (Cadesky *et al.* 2000).

A meta-analysis of the existing literature can provide the opportunity to reveal the specific nature of social cognitive deficits in ADHD, including at different stages of development and can have important implications for improving social functioning in ADHD. In this meta-analysis, the main goal was to investigate social cognition in ADHD. In this meta-analysis, we compared emotion recognition and ToM deficits in ADHD with healthy controls and ASD by taking into account the relationship of social cognitive deficits with important confounders including age, co-morbidity, ADHD subtypes and intellectual abilities. We hypothesized that ADHD would be associated with significant impairment (but less severe than observed in ASD) in social cognition in comparison with healthy controls.

Method

Study selection

PRISMA (preferred reporting items for systematic reviews and meta-analyses) guidelines were used in conducting this meta-analysis (Moher *et al.* 2009). A literature search was conducted using the databases PubMed, PsycINFO and Scopus to identify the relevant studies (January 1987 to June 2015) using the combination of key words as follows: ADHD, emotion recognition, theory of mind and social cognition. Reference lists of published reports were also reviewed for additional studies. Inclusion criteria were studies that: (1) investigated emotion recognition or ToM abilities; (2) reported sufficient data to calculate the effect size and standard error of the emotion recognition or ToM measures including results of parametric statistics (i.e. *t* and *F* values); (3) compared the performances of patients with ADHD and healthy controls or ASD (according to DSM-IV, DSM-III-R or similar criteria).

The selection process is summarized in online Supplementary Fig. S1. We contacted three authors of papers that did not report sufficient data to calculate effect sizes in their published abstracts and received relevant data from two of them. Studies investigating emotion recognition abilities with methods other than facial and vocal recognition were excluded as only a few studies used such methods (i.e. body language, scenes). After an initial screening of articles retrieved by literature search, 84 articles were reviewed. Of these, 52 articles met our inclusion criteria. Four studies were excluded as they overlapped with other studies included (studies with most up-to-date, larger sample size were selected). A total of 48 studies were included in the meta-analysis (Table 1).

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Table 1. *Studies included in the meta-analysis of social cognition in ADHD in comparison with HC and ASD*

Studies	Sample	IQ	Social cognition	Characteristics	Co-morbidity	Social cognition outcome
Adachi *et al.* (2004)	29 ADHD, 54 ASD, 199 HC	WISC	Sarcasm	Age (ADHD) = 9.6 years Age (ASD) = 9.8 years All ADHD C	MR excluded	Only HFPDD impaired
Albiez (2014)	33 ADHD, 33 HC		RMET MASC	Age (ADHD) = 37.6 years 26 C, 7 I	Psychosis, substance use excluded	No difference
Allen (2010)	10 ADHD, 4 HC 12, ASD	WASI	RMET	Age (ADHD) = 9.0 years Age (ASD) = 10.7 years	Other Axis I excluded	Not impaired
Altıntas (2014)	30 ADHD, 30 HC		Hinting *Faux pas* FB 1 and 2	Age (ADHD) = 25.7 years	MR, psychosis excluded	No difference
Aspan *et al.* (2014)	22 ADHD, 22 HC		Face emotion Ekman 60	Age (ADHD) = 14.2 years All C No medication 72 h	ASD, substance use, MR excluded 3/22 CD, 2/22 OCD	ADHD impaired in fear and more sensitive to disgust
Ayaz *et al.* (2013)	64 ADHD, 69 HC	WISC	RMET	Age (ADHD) = 13.2 years No medication 24 h	MR, ASD excluded	ADHD impaired
Azadi Sohi (2012)	20 ADHD, 19 HC, 13 ASD	WISC	Happé stories	Age (ADHD) = 11.2 years Age (ASD) = 10.9 years	MR, CD excluded	ADHD impaired
Bagcioglu *et al.* (2014)	34 ADHD, 39 HC		Face emotion Ekman	Age (ADHD) = 22.0 years No medications	Axis I excluded All antisocial personality	No difference
Baribeau *et al.* (2015)	71 ADHD, 34 HC, 118 ASD	WISC, WASI, Stanford-Binet	RMET	Age (ADHD) = 9.3 years Age (ASD) = 11.6 years		ADHD impaired ASD more impaired
Bean (2013)	13 ADHD, 14 HC, 19 ASD	Stanford-Binet	NEPSY ToM	Age (ADHD) = 12.5 years Age (ASD) = 13.3 years		No difference
Boakes *et al.* (2008)	24 ADHD, 24 HC	WISC	Face emotion-photographs and videos	Age (ADHD) = 10.2 years >20 h no medication	MR excluded	ADHD impaired in disgust and fear

Table 1 (*cont.*)

Studies	Sample	IQ	Social cognition	Characteristics	Co-morbidity	Social cognition outcome
Bühler *et al.* (2011)	84 ADHD, 86 ASD	WISC	Face emotion Adapted from Ekman pictures	Age (ADHD) = 9.7 years Age (ASD) = 10.8 years	Axis I excluded Subgroup with CD	ASD marginally more impaired
Buitelaar *et al.* (1999)	9 ADHD, 20 HC, 18 ASD	WISC	Face emotion Ekman ToM 1 ToM 2	Age (ASD) = 12.4 years No medications		No significant difference between ADHD and ASD
Cadesky *et al.* (2000)	149 ADHD, 27 HC	WISC	Face emotion DANVA	Age (ADHD) = 9.1 years No medication 24 h	Anxiety/depression MR excluded 63 CD	ADHD is impaired
Caillies *et al.* (2014)	15 ADHD, 15 HC		ToM-FB2 Sarcasm	Age (ADHD) = 9.0 years No medication 24 h 3 I, 8 C, 4 H/I types	No mention	ADHD impaired
Charman *et al.* (2001)	22 ADHD, 22 HC		ToM Happé stories	Age (ADHD) = 9.7 years All C type No medication 24 h Both DSM-ICD	CD excluded	No difference
Corbett & Glidden (2000)	37 ADHD, 37 HC		Face emotion Ekman Vocal emotion	Age (ADHD) = 10.1 years All C type No medication 18–24 h	Learning disorder, MR, CD excluded	
Da Fonseca *et al.* (2009)	27 ADHD, 27 HC	WISC (only ADHD)	Face emotion Photographs	Age (ADHD) = 10.2 years All C type No medication 24 h	6 ODD, 1 CD No MR	ADHD impaired
Demopoulos *et al.* (2013)	115 ASD, 276 ADHD	WISC	Face emotion DANVA Vocal emotion	Children		ASD more impaired
Demurie *et al.* (2011)	13 ADHD, 13 HC, 13 ASD	WISC	RMET	Age (ADHD) = 13.7 years Age (ASD) = 14.35 years	MR	

Downs & Smith (2004)	16 ADHD, 10 HC, 10 ASD	WISC	Face emotion Pictures and drawings	Age (ADHD) = 8.3 years Age (ASD) = 7.1 years	All ADHD-ODD No other co-morbidity	ASD more impaired
Dyck *et al.* (2001)	35 ADHD, 36 HC, 48 ASD	WISC	ToM Strange stories Face emotion Photographs	All I type ADHD		Only ASD is impaired in ToM. Both patient groups impaired in emotion recognition
Gonzalez-Gadea *et al.* (2013)	22 ADHD, 23 ASD, 21 HC		*Faux pas* RMET	Age (ADHD) = 35.3 years Age (ASD) = 33.0 years 55% stimulants	Axis I excluded	ToM impaired in AS
Greenbaum *et al.* (2009)	30 ADHD, 34 HC	WASI	False belief Interpretative ToM Face emotion MNTAP	Age (ADHD) = 9.3 years Majority C type 18 on medication	7 Learning disability, 3 ODD, 2 anxiety or depression	No difference
Ibáñez *et al.* (2014)	16 ADHD, 41 HC		*Faux pas* RMET	Age (ADHD) = 34.6 years		ADHD impaired in RMET
Kuijper *et al.* (2015)	37 ADHD, 38 HC, 46 ASD	WISC	FB1 and FB2	Age (ADHD = 8.9 years Age (ASD) = 9.3 years 19 C, 12 H/I, 6 I No medication		ADHD impaired No difference between ASD and HC
Lee *et al.* (2009)	42 ADHD, 45 HC		Face emotion Matsumoto-Ekman	Age (ADHD) = 8 years 9 I, 29 C, 4 HI type 12 on medication	ASD, MR excluded	No difference Inattentive less well performance
Mary *et al.* (2015)	31 ADHD, 31 HC	WISC	*Faux pas* RMET	Age (ADHD) = 10.3 years 28 C, 3 I type 14 on medications–24 h off	Autism, MR excluded	ADHD impaired in both ToM tasks
Miller *et al.* (2011)	33 ADHD, 18 HC	WAIS	Face emotion DANVA	Age (ADHD) = 35.0 years 17 C, 16 I 24 h medication off	MR excluded 5 LD, 5 depression/dysthymia	Only inattentive correlated
Muris *et al.* (1999)	14 ADHD, 20 ASD	WISC	FB1 and FB2	Age (ADHD) = 8.5 years Age (ASD) = 9.3 years		ASD impaired in comparison with ADHD
Norman (1999)	17 ADHD, 20 HC		False belief Happé stories	Age (ADHD) = 10.1 years		

Table 1 (*cont.*)

Studies	Sample	IQ	Social cognition	Characteristics	Co-morbidity	Social cognition outcome
Nydén *et al.* (2010)	73 ADHD, 55 ASD	WAIS	Happé cartoons	Age (ADHD) = 33.2 years Age (ASD) = 32 years 42 C, 22 I, 9 H/I	LD, MR excluded	No difference
Pelc *et al.* (2006)	30 ADHD, 30 HC		Face emotion Morphing	Age (ADHD) = 8 years 22 on medications but off >10 days	No ASD, CD, LD, MR	Anger and sadness impaired
Rapport *et al.* (2002)	28 ADHD, 28 HC	WAIS	Face emotion Vocal emotion DANVA	Age (ADHD) = 36.3 years	Substance use excluded	Fear, happiness, anger impaired
Retz-Junginger *et al.* (2015)	138 ADHD, 40 HC		Face emotion	Age (ADHD) = 31.8 years 91 C	ASD, MR, other co-morbidities excluded	Negative emotions impaired
Saeedi *et al.* (2014)	30 ADHD, 30 HC		RMET	Age (ADHD) = 8.3 years No medications	MR excluded	ADHD impaired
Schönenberg *et al.* (2015)	25 ADHD, 25 HC		Face emotion Morphing	Age (ADHD) = 34.8 years All C type	MR, substance use, Axis I excluded	Fear and sadness impaired
Schwenck *et al.* (2013)	56 ADHD, 28 HC	WISC	Face emotion Morphing	Age (ADHD) = 12.4 years 44 I, 10 C, 2 H/I No medication 24 h	CD, ASD excluded	No difference
Seymour *et al.* (2013)	38 ADHD, 41 HC	WASI	Face emotion DANVA	Age (ADHD) = 12.1 years	MR, mood/anxiety disorders excluded 8 ODD	
Shapiro *et al.* (1993)	67 ADHD, 38 HC	Kaufman brief IQ	Face emotion MNTAP	Age (ADHD) = 8.9 years	Not clear	No difference
Shuai *et al.* (2011)	375 ADHD, 125 HC		False belief	Age = 10.0 years 200 I, 150 C, 25 H/I	55 ODD/CD 39 LD	ADHD impaired
Sinzig *et al.* (2008)	30 ADHD, 29 HC, 40 ASD	WISC	Face emotion Frankfurt test	Age (ADHD) = 8.5 years Age (ASD) = 9.3 years 17 I, 13 C No medication 24 h	CD 53% MR excluded No co-morbid disorders	ADHD impaired

Sjöwall *et al.* (2013)	102 ADHD, 102 HC	Face emotion NimStim	71 C, 27 I, 4 H/I 10 took medication last 24 h	CD or ODD 46% Anxiety disorder 8% MR excluded	Impaired in all but disgust
Sodian & Hülsken (2005)	32 ADHD, 101 HC	FB2, Happé stories Epistemic state attribution task	Age (ADHD) = 8.9 years	No mention	Impaired only in attribution task
Tatar *et al.* (2014)	40 ADHD, 40 HC	Face emotion Ekman	Age (ADHD) = 26 years 35/40 I, 5 C	14 anxiety disorders or mild depression Other Axis I and substance abuse excluded	ADHD impaired
Williams *et al.* (2008)	51 ADHD, 51 HC	Face emotion-photographs	Age (ADHD) = 13.8 years	MR and substance use disorders excluded	ADHD impaired in anger and fear
Yang *et al.* (2009)	26 ADHD, 30 HC, 20 ASD	ToM-false belief	Age (ADHD) = 13.5 years Age (ASD) = 15.5 years 3 I, 17 C	No mention	ADHD-not different from controls ASD more impaired
Yılmaz (2014)	30 ADHD, 30 HC	RMET	Age (ADHD) = 25.6 years No medication 48 h	MR and active Axis I and substance use excluded	No difference

ADHD, Attention-deficit/hyperactivity disorder; HC, healthy controls; ASD, autism spectrum disorder; IQ, intelligence quotient; WISC, Wechsler Intelligence Scale for Children; C, combined type; MR, mental retardation; HFPDD, high-functioning pervasive developmental disorder; RMET, Reading the Mind from Eyes Test; MASC, Movie for the Assessment of Social Cognition; I, inattentive type; WASI, Wechsler Abbreviated Scale of Intelligence; FB, False belief task; CD, conduct disorder; OCD, obsessive–compulsive disorder; NEPSY, A Developmental NEuroPSYchological Assessment; ToM, theory of mind; DANVA, Diagnostic Analysis of Nonverbal Accuracy; H/I, hyperactive impulsive type; DSM, Diagnostic and Statistical Manual of Mental Disorders; ICD, International Classification of Diseases; ODD, oppositional defiant disorder; AS, Asperger's syndrome; MNTAP, Minnesota Test of Affective Processing; FB1, first-order FB; FB2, second-order FB;WAIS, Wechsler Adult Intelligence Scale; LD, learning disability.

ADHD v. *healthy controls*

A total of 44 studies (three of the studies have two ADHD samples) comparing ADHD subjects ($n = 1998$) (78.8% male) and healthy controls ($n = 1725$) (64.9% male) were included in the study. There was a significantly higher ratio of males in ADHD in comparison with the control group [relative risk (RR) = 1.24, 95% confidence interval (CI) 1.14–1.34, $Z = 4.9$, $p < 0.001$]. There was no significant between-group difference for age ($d = 0.03$, 95% CI −0.12 to 0.18, $Z = 0.40$, $p = 0.69$). Intelligence quotient (IQ) score was significantly lower in ADHD patients in comparison with healthy controls ($d = 0.61$, 95% CI 0.42–0.80, $Z = 6.39$, $p < 0.001$). Ages of the ADHD group ranged between 8 and 36.3 years and clustered as studies in children (mean age range = 8–15 years) and adults (mean age range = 22–37 years). In the studies that gave information about ADHD subtype ($n = 1089$), combined type ($n = 662$) followed by inattentive type ($n = 373$) was most common and very few individuals ($n = 54$) had a diagnosis of ADHD of the hyperactive/impulsive subtype. ADHD in adulthood was diagnosed at the time of the study (retrospectively).

ADHD v. *ASD*

A total of 17 studies (two of the studies have two ASD samples) consisting of 772 ADHD (80.1% males) and 710 ASD (84.0% males) patients were included in the meta-analysis. There was no significant between-group difference for the ratio of males (RR = 1.03, 95% CI 0.98–1.08, $Z = 1.15$, $p = 0.25$) or age ($d = 0.11$, 95% CI −0.11 to 0.32, $Z = 0.97$, $p = 0.33$). There were no significant IQ differences between ADHD and ASD ($d = 0.01$, 95% CI −0.28 to 0.30, $Z = 0.09$, $p = 0.93$). Only two adult studies investigated differences between ASD and ADHD. The mean age range for childhood studies was 7 to 15 years.

Social cognition measures

Studies have investigated facial emotion recognition with a variety of methods [different sets of images of faces to label: most commonly Ekman and Diagnostic Analysis of Nonverbal Accuracy (DANVA)] (Ekman & Friesen, 1976; Nowicki & Carton, 1993). Different ToM tasks have been utilized across studies, most commonly the Reading the Mind in the Eyes Task (RMET) and Happé stories; other tasks included different versions of false belief and intention tasks, recognition of *faux pax* and sarcasm (Happé, 1994; Baron-Cohen *et al.* 2001).

Statistical analyses

Effect sizes for total scores for facial and vocal emotion recognition and individual effect sizes for facial recognition of six basic emotions (happy, sad, anger, disgust, fear and surprise) were calculated. For studies that did not report total scores but only scores for individual emotions, a pooled score of the facial emotion recognition score was calculated. For studies that reported more than one ToM task, a pooled effect size and standard error were calculated. Individual meta-analyses for individual ToM tasks that are reported by at least five studies (RMET, Happé stories) were also conducted. Finally, a mean score for social cognition was calculated for studies reporting both ToM and emotion recognition. Meta-analyses were performed using packages in the R environment (OpenMetaAnalyst, Metafor) and MIX software version 1.7 on a Windows platform (Bax *et al.* 2006; Viechtbauer, 2010; Wallace *et al.* 2012). Effect sizes were weighted using the inverse variance method and a random-effects model (DerSimonian–Laird estimate) was used because the distributions of effect sizes were heterogeneous for the number of variables. The magnitude of effect sizes was interpreted based on guidelines [$d = 0.2$–0.5 (small), $d = 0.5$–0.8 (medium), $d > 0.8$ (large)] suggested by Cohen (1988). Homogeneity of the distribution of weighted effect sizes was tested with the I^2 and Q-tests. The I^2 statistic describes the percentage of variation across studies that is due to heterogeneity rather than chance and the Q-test is the weighted sum of squared differences between individual study effects and the pooled effect across studies. A significant Q-test suggests that the distribution of effect sizes is not homogeneous (Lipsey & Wilson, 2001). Tau squared (τ^2), an estimate of between-study variance, was used as a measure of the magnitude of heterogeneity in the random-effects model. The possibility of publication bias was assessed with Egger's test. The fail-safe N test was also used. This test involves computing a combined p value for all studies included in the meta-analysis, and calculating how many additional studies with a zero effect (average Z of zero) would be necessary to create a non-significant p value.

Meta-regression analyses were conducted for variables that are reported by at least 10 samples. These variables included age, gender, ADHD subtype (percentage of individuals with ADHD that met criteria for combined type), co-morbidity (ratio of patients who also met criteria for conduct disorder) and IQ (effect sizes of patient–control differences). Pre-morbid IQ measures and measures only assessing performance IQ (Raven's progressive matrices and Gong's non-verbal IQ) were not included as IQ measures (see Table 1). Meta-regression analyses performed with a random-effects model were conducted using the restricted-information maximum likelihood method with a significance level set at $p < 0.05$. Subgroup analyses were

Table 2. *Mean weighted effect sizes for social cognition between ADHD and healthy controls*

Test	Sample	ADHD	Healthy controls	*d*	95% CI	*Z*	*p*	*Q*-test *p*	τ^2	Bias
Social cognition	47	1999	1725	0.44	0.33 to 0.55	7.94	<0.001	<0.001	0.07	0.32
Child	35	1570	1380	0.52	0.40 to 0.64	8.24	<0.001	<0.001	0.07	
Adult	12	429	345	0.22	0.04 to 0.40	2.43	0.02	0.003	0.03	
Facial emotion	25	1021	764	0.44	0.30 to 0.59	6.04	<0.001	0.001	0.07	0.70
Child	18	723	574	0.49	0.30 to 0.68	5.16	<0.001	<0.001	0.10	
Adult	7	298	190	0.33	0.14 to 0.52	3.48	0.001	0.53	0	
Total score	15	581	430	0.51	0.29 to 0.73	4.56	<0.001	<0.001	0.13	0.54
Anger	15	818	702	0.48	0.28 to 0.68	4.73	<0.001	<0.001	0.09	0.65
Disgust	13	729	610	0.17	−0.01 to 0.35	1.86	0.06	0.03	0.05	0.81
Fear	14	788	672	0.47	0.30 to 0.64	5.44	<0.001	0.03	0.05	0.61
Sad	15	818	702	0.24	0.13 to 0.36	4.05	<0.001	0.54	0	0.77
Happy	14	788	672	0.22	0.06 to 0.38	2.74	0.006	0.06	0.04	0.90
Surprise	9	519	428	0.27	0.09 to 0.46	2.90	0.004	0.17	0.02	0.79
Vocal emotion	6	247	110	0.40	0.05 to 0.76	2.24	0.02	0.02	0.12	0.35
ToM	24	1010	1024	0.45	0.29 to 0.62	5.37	<0.001	<0.001	0.09	0.26
Child	19	879	869	0.56	0.40 to 0.72	6.92	<0.001	0.02	0.05	
Adult	5	131	155	0.04	−0.24 to 0.33	0.30	0.76	0.21	0.03	
RMET	10	320	311	0.44	0.10 to 0.77	2.56	0.01	<0.001	0.21	0.63
Happé stories	5	116	198	0.51	0.15 to 0.88	2.75	0.006	0.07	0.09	0.35

ADHD, Attention-deficit/hyperactivity disorder; *d*, effect size of between-group difference; CI, confidence interval; bias, *p* value of Egger's test; ToM, theory of mind; RMET, Reading the Mind in the Eyes Test.

done for adulthood and childhood ADHD. The Q_{bet} test was used to compare these subgroups.

Results

ADHD v. *healthy controls*

Social cognition was significantly impaired ($d = 0.44$) in ADHD patients in comparison with healthy controls (Table 2). When social cognitive domains were investigated separately, ADHD subjects performed significantly more poorly than healthy controls in ToM ($d = 0.45$) and facial ($d = 0.44$) and vocal emotion recognition ($d = 0.40$) (Table 2). There was a significant heterogeneity for distributions of effect sizes for all these social cognition domains ($I^2 = 33$–59% and p for Q-tests $\leqslant 0.001$) but magnitudes of heterogeneity were modest ($\tau^2 = 0.07$–0.09) in the random-effects model. There was no evidence of publication bias and fail-safe N values for social cognition measures were very large ($n = 454$–1734).

In recognition of individual facial emotions, the most significant impairments were evident in anger ($d = 0.49$) and fear ($d = 0.48$). In ADHD, recognition of sad ($d = 0.24$), surprised ($d = 0.27$) and happy ($d = 0.22$) faces were also mildly but significantly impaired. There was no significant between-group difference for recognition of disgust. In individual ToM measures, ADHD was associated with poorer performance than controls for both RMET ($d = 0.44$) and Happé stories ($d = 0.51$) tasks. Distributions of effect sizes for RMET, Happé stories, recognition of anger, fear, disgust and happy were significantly heterogeneous (Table 2). However, magnitudes of heterogeneous distributions were modest in the random-effects model ($\tau^2 = 0$–0.21). There was no evidence of publication bias for any of the emotion recognition or individual ToM measures. In a subgroup analysis of studies that explicitly stated ASD co-morbidity as an exclusion criteria, ADHD patients significantly underperformed healthy controls ($d = 0.37$, 95% CI 0.11–0.63, $Z = 2.75$, $p = 0.006$).

Meta-regression analyses

Deficits in social cognition score in ADHD were not significantly related to gender ($Z = 0.29$, $p = 0.97$, $k = 39$), age ($Z = -1.95$, $p = 0.05$, $k = 45$), percentage of individuals with the combined type of ADHD ($Z = 0.31$, $p = 0.75$, $k = 25$), or with co-morbid conduct disorder ($Z = -0.41$, $p = 0.68$, $k = 12$). When meta-regression analyses for age were conducted separately for ToM ($Z = -2.83$, $p = 0.005$) and facial emotion recognition ($Z = 0.33$, $p = 0.74$), younger age in the ADHD group was associated with more severe ToM but not emotion recognition deficits.

IQ differences between ADHD subjects and healthy controls were significantly associated with more severe

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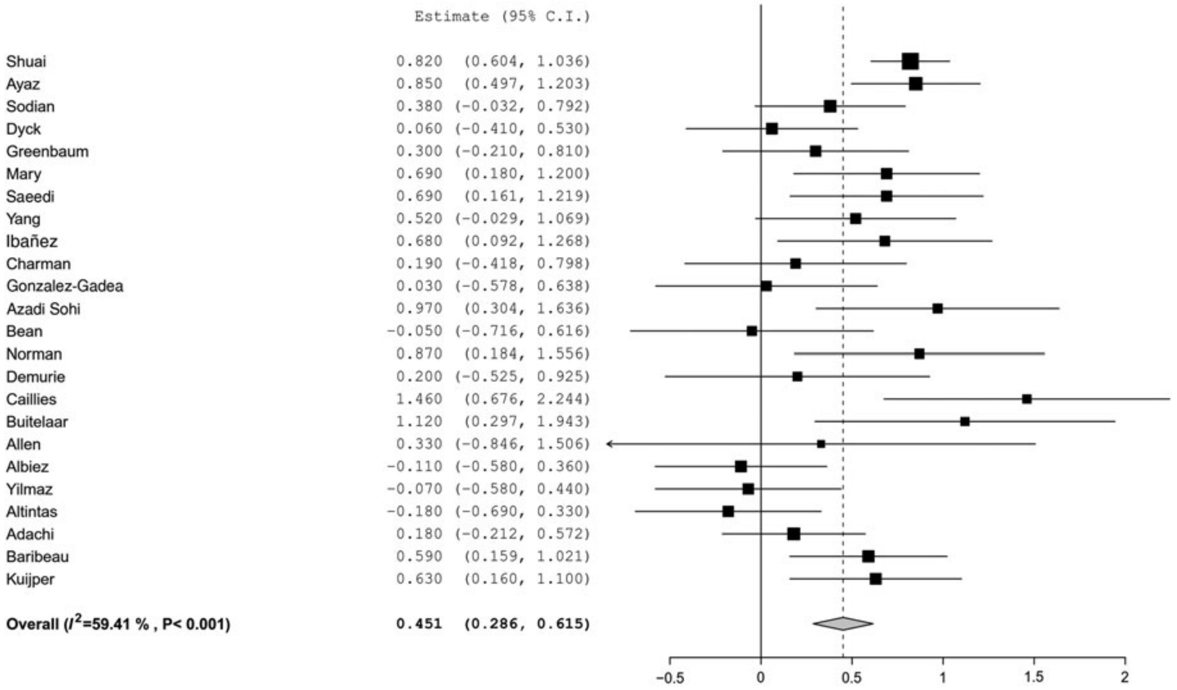

Fig. 1. Forest plot for theory of mind differences between attention-deficit/hyperactivity disorder patients and healthy controls. C.I., Confidence interval.

social cognitive impairment ($Z = 2.45$, $p = 0.01$, $k = 24$). However, impairment in social cognition was also evident in samples matched for IQ ($d = 0.37$, 95% CI 0.16–0.59, $Z = 3.4$, $p < 0.001$, $k = 12$).

Childhood v. *adulthood ADHD*

Impairment in social cognition in ADHD was significantly more severe in the child than adult samples ($d = 0.52$ *v.* 0.22, $Q_{\text{bet}} = 15.3$, $p < 0.001$). Among social cognition domains, differences between child and adult samples were even more pronounced for ToM ($d = 0.56$ *v.* 0.04, $Q_{\text{bet}} = 18.0$, $p < 0.001$), with no evidence of ToM impairments in ADHD individuals in adulthood (Fig. 1). Differences between child and adult samples were not significant for facial emotion recognition ($d = 0.49$ *v.* 0.33, $Q_{\text{bet}} = 1.72$, $p = 0.19$) (Fig. 2).

ADHD v. *ASD*

Overall social cognition was significantly impaired ($d = 0.54$) in ASD in comparison with ADHD subjects. ToM impairment (Fig. 3) was particularly more severe in ASD than ADHD patients ($d = 0.77$) (Table 3). The between-group difference for facial emotion recognition was small ($d = 0.34$) but significant (Fig. 4) (Cohen, 1988). There was no evidence of publication bias and the fail-safe N values for social cognition measures were large ($n = 30$–374). There was significant heterogeneity in the distribution of effects sizes of ToM ($\tau^2 = 0.14$–0.31, $I^2 = 72$–79%, p for Q tests $\leqslant 0.001$).

In meta-regression analyses, IQ ($Z = 4.43$, $p < 0.0001$) but not age ($Z = 0.15$, $p = 0.88$, $k = 15$) and gender ($Z = 0.06$, $p = 0.96$) had significant effects on between-group differences for social cognition scores. However, in subgroup analyses of studies matched for IQ, differences between ASD and ADHD were relatively smaller but still significant for ToM ($d = 0.52$) and overall social cognition score ($d = 0.36$) but not for facial emotion recognition ($d = 0.10$). The distribution of effect sizes of all of these measures was homogeneous in IQ-matched samples ($\tau^2 = 0.04$–0.05, p for $Q > 0.05$).

Discussion

The current meta-analysis investigated social cognition in ADHD patients in comparison with healthy controls and ASD subjects. Emotion recognition and ToM were significantly impaired in ADHD relative to control subjects. In facial emotion recognition, the most severe deficits were evident in recognition of anger and fear. The severity of social cognitive impairment in ADHD subjects was modest and social cognitive deficits were less pronounced in adulthood. ToM deficits were significantly less severe in ADHD compared with ASD patients, even after accounting for the effects of IQ.

ADHD was associated with deficits with medium effect sizes in ToM and emotion recognition. The severity

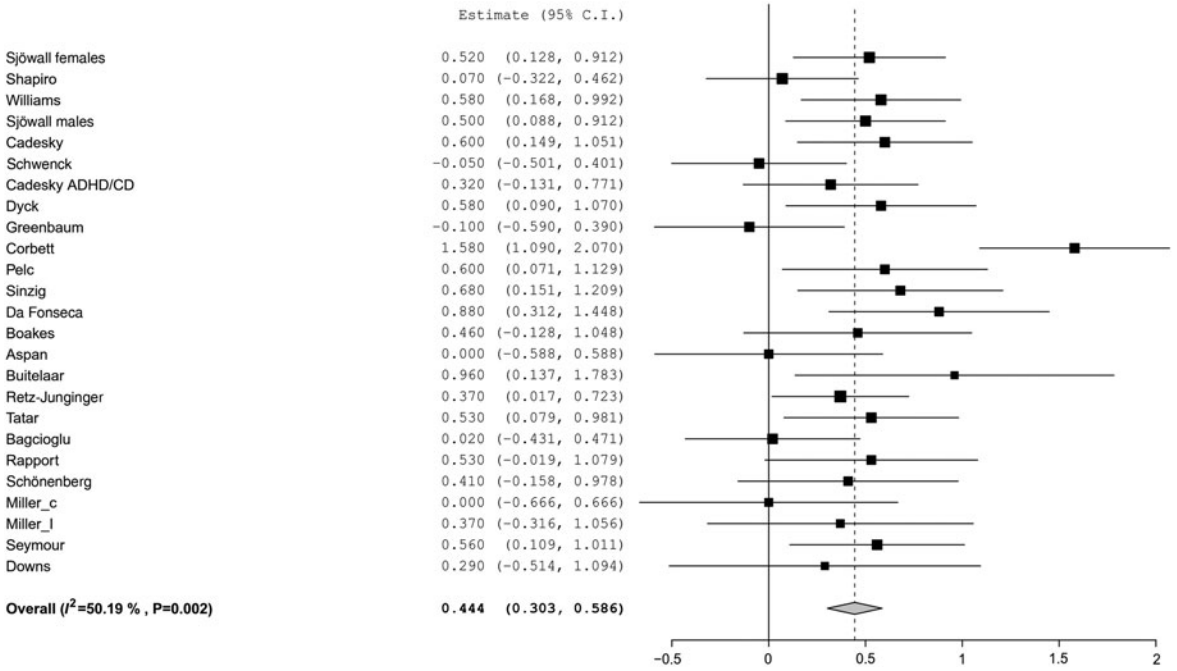

Fig. 2. Forest plot for facial emotion recognition differences between attention-deficit/hyperactivity disorder (ADHD) patients and healthy controls. C.I., Confidence interval; CD, conduct disorder.

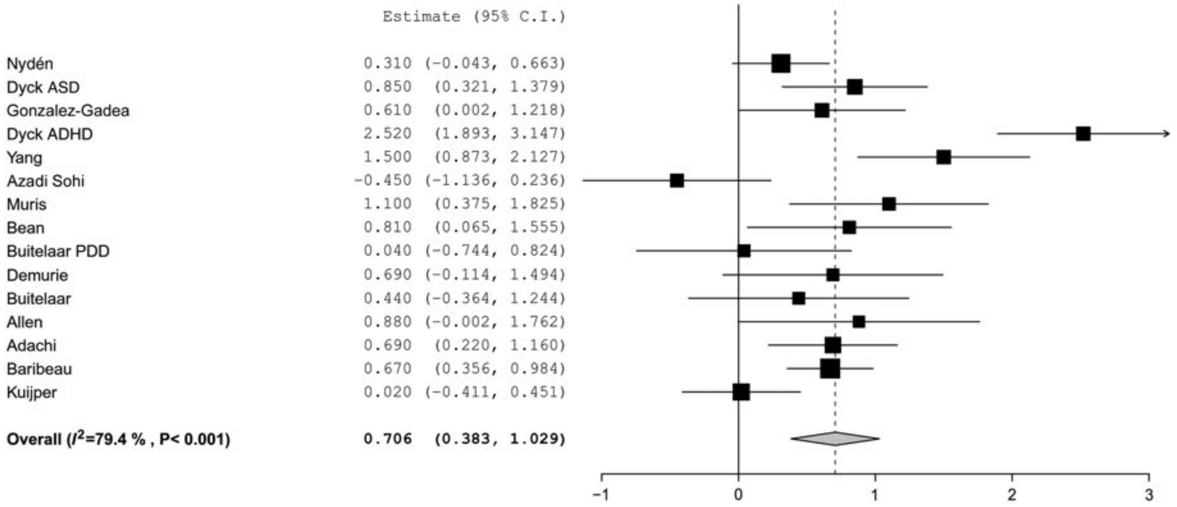

Fig. 3. Forest plot for theory of mind differences between autism spectrum disorders (ASD) and attention-deficit/hyperactivity disorder (ADHD). C.I., Confidence interval; PDD, pervasive developmental disorder.

of social cognitive impairment in ADHD patients was comparable with other neurocognitive deficits including verbal memory and most measures of attention and executive functions. Only some aspects of executive functions (i.e. response inhibition) and attention (sustained attention) might be relatively more severely impaired than social cognition in ADHD (Boonstra *et al.* 2005; Schoechlin & Engel, 2005; Willcutt *et al.* 2005; Bálint *et al.* 2009). These findings suggest that a sizeable minority of patients with ADHD have significant difficulties in social cognitive abilities. Interestingly, emotion recognition deficits were relatively specific to anger and fear. This finding is in accordance with findings suggesting abnormal electrophysiological and disrupted temporal lobe activity in response to angry faces compared with happiness and sadness in ADHD (Ichikawa *et al.* 2014; Köchel *et al.* 2014). Difficulties in recognition of anger and fear in others and impairments

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Table 3. *Mean weighted effect sizes for social cognition between ASD and ADHD*

Test	Sample	ASD	ADHD	*d*	95% CI	Z	*p*	*Q*-test *p*	τ^2	Bias
Social cognition	19	710	772	0.54	0.32 to 0.76	4.82	<0.001	<0.001	0.15	0.64
IQ matched	14	377	473	0.36	0.15 to 0.56	3.44	<0.001	0.09	0.05	
Facial emotion	8	317	444	0.34	0.01 to 0.67	2.00	0.04	<0.001	0.14	0.61
IQ matched	6	182	157	0.10	−0.18 to 0.38	0.67	0.50	0.21	0.04	
ToM	15	459	372	0.71	0.38 to 1.03	4.26	<0.001	<0.001	0.31	0.47
IQ matched	9	281	253	0.52	0.22 to 0.77	4.06	<0.001	0.13	0.05	

ASD, Autism spectrum disorders; ADHD, attention-deficit/hyperactivity disorder; *d*, effect size of between-group difference; CI, confidence interval; bias, *p* value of Egger's test; IQ, intelligence quotient; ToM, theory of mind.

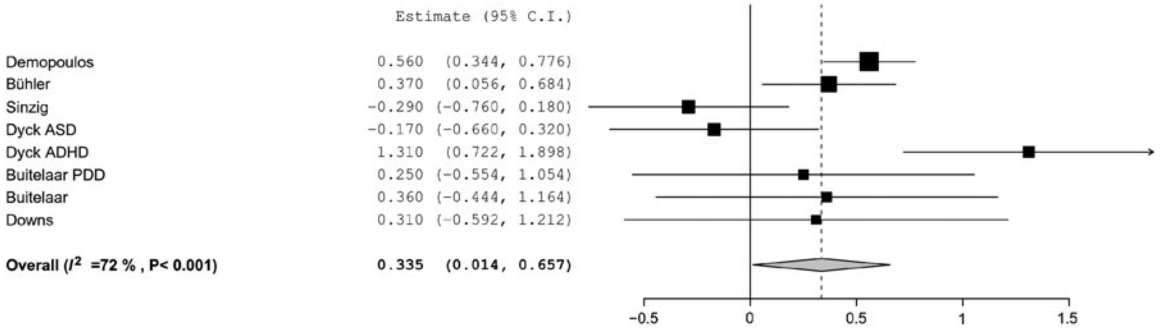

Fig. 4. Forest plot for facial emotion recognition differences between autism spectrum disorders (ASD) and attention-deficit/hyperactivity disorder (ADHD). C.I., Confidence interval; PDD, pervasive developmental disorder.

in more complex aspects of social cognition can contribute to the behavioural and interpersonal problems observed in ADHD. Interventions targeting social cognitive deficits in ADHD patients can potentially improve functioning in ADHD. There is preliminary evidence suggesting that stimulants can improve social cognitive abilities in ADHD, but there is a need for further studies to confirm these early findings (Maoz *et al.* 2014). A number of studies have suggested that cognitive training might improve neurocognitive deficits in ADHD; future studies should consider incorporating social cognition in training programmes to improve social functioning in ADHD (Cortese *et al.* 2015).

Social cognition deficits, especially in ToM, were significantly more pronounced in ASD in comparison with ADHD subjects. This finding is in accordance with the outcome of previous meta-analyses of social cognitive deficits in ASD patients in comparison with healthy controls (Yirmiya *et al.* 1998; Uljarevic & Hamilton, 2013; Lozier *et al.* 2014), which reported larger effect sizes compared with our meta-analysis findings comparing ADHD and controls. ASD-specific abnormalities in brain structure can contribute to social cognitive differences between ASD and ADHD (Lim *et al.* 2015). However, the magnitude of differences, even for ToM, suggests that there is considerable overlap between groups and social cognitive deficits cannot be considered to be specific to ASD. Some of the studies included have excluded co-morbid ASD and other Axis I disorders (Table 1). In studies that explicitly stated co-morbid ASD as an exclusion criteria, social cognitive impairment was still significant ($d = 0.37$). However, many of these patients might still have subthreshold autistic traits (Taylor *et al.* 2013). There is a need for studies investigating the effect of autistic traits in ADHD on social cognition.

Another important point is the heterogeneity of ADHD patients including in their neurocognitive profile (van Hulst *et al.* 2015). One of the studies included in the meta-analysis compared ToM abilities of three subtypes of ADHD and did not find any difference (Shuai *et al.* 2011). There was no significant effect of the ratio of inattentive and combined subtypes of ADHD on social cognition in the current meta-analysis. This finding suggests that social cognitive abilities do not differ between these two subtypes but this finding needs to be confirmed by studies directly comparing these subtypes. Also, an only small minority of individuals with ADHD had hyperactive/impulsive type in this meta-analysis. Therefore, there is a need for further studies exploring social cognition in subtypes of ADHD. Our meta-analysis has suggested that social cognitive deficits in ADHD were not related to having co-morbid conduct disorder. ADHD symptoms are also associated with other neurodevelopmental disorders including learning disorders and can precede adult-onset neurodevelopmental disorders (Semrud-Clikeman *et al.* 1992; Peralta *et al.* 2011). Recent familial and genetic studies in ADHD also found evidence of partial overlap with autism, neurodevelopmental disorders, schizophrenia and bipolar disorder (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013). All of these disorders are associated with significant social impairment and social cognitive deficits (Yirmiya *et al.* 1998; Bora *et al.* 2005; Bora & Pantelis, 2013). It can be hypothesized that in some patients with a diagnosis of ADHD, inattention, impulsivity and hyperactivity symptoms might be markers of more widespread neurodevelopmental problems, neuroanatomical abnormalities and neurocognitive deficits (including social cognition) rather than reflecting a more limited developmental abnormality associated with problems in higher-order functional organization [i.e. maturational delay in default mode network and its connections (Bush, 2010; Sripada *et al.* 2014)]. Identifying patients with broad neurodevelopmental problems is important as these individuals might have a higher risk of developing adult-onset disorders (such as psychotic disorders) and can have substantial impairment in social functioning in adulthood.

Our findings suggest that age plays an important role in the differences found between ADHD and controls. In paediatric samples, deficits with medium effect sizes were observed in social cognition. As a limitation, there was no information available for the deficit in social cognition in adolescent and young adults with ADHD (mean age 15–22 years). However, in adulthood, the effect size of the social cognitive deficit was small and was non-significant for ToM. This finding cannot be considered as a ceiling effect as adult ADHD studies have mainly used advanced ToM tasks that were sensitive to ToM impairment in psychiatric disorders such as bipolar disorder (Bora *et al.* 2015). Potential age-related improvements in ADHD have been also reported for other neurocognitive abilities (Coghill *et al.* 2014). These findings support the views that consider ADHD as a disorder characterized by delay in neurodevelopment of high-order functions in most patients (Shaw *et al.* 2007; Sripada *et al.* 2014); it seems that performances of these individuals with ADHD catch up with non-ADHD subjects as they get older. A delayed developmental trajectory for acquisition of social cognitive abilities in ADHD can also explain more pronounced problems in recognition of anger and fear, as the ability to recognize these emotions fully develops later than for happiness and sadness (Boyatzis *et al.* 1993). However, it is also important to consider the possibility that individuals with adult ADHD perform better in social cognitive tasks because many individuals with 'adult ADHD' might be misdiagnosed with a childhood neurodevelopmental disorder based on self-report and do not have 'true ADHD' (Moffitt *et al.* 2015). It is also important to note that clinically, the developmental trajectory of most individuals with ADHD differs from ASD. The developmental trajectory of ASD might be characterized by increasingly falling behind the trajectory of a healthy population in some cognitive domains. Thus, the rate of improvement in a healthy population (typically developing children and adolescents) may accelerate faster that for ASD, given the findings of another meta-analysis suggesting that difficulties in facial emotion recognition can actually increase with age in ASD (Lozier *et al.* 2014). It is important to increase our efforts to map the trajectories of ADHD and other childhood-onset psychiatric disorders and investigate relationships between abnormal cortical development and the trajectory of symptoms and cognitive deficits during development (Shaw *et al.* 2013; Cropley & Pantelis, 2014; Giedd *et al.* 2015; Zalesky *et al.* 2015). Better definition of the longitudinal course of broad categories such as ADHD can help us to characterize more valid subtypes of neurodevelopmental disorders and improve our management strategies.

Another important consideration is the effect of neurocognition on ADHD–healthy control and ADHD–ASD differences. Our findings have suggested that IQ was a significant confounder for between-group differences in social cognition. There were more differences between ASD and ADHD on social cognition tasks when IQ was significantly lower in ASD in comparison with ADHD. IQ has also played a significant role in comparison of group differences between ADHD and controls in social cognition tasks. However, even in IQ-matched groups, there was a significant difference in the severity of social cognitive impairment between ADHD and ASD and controls. These findings suggest that more general cognitive impairment can partially account for group differences between ADHD and ASD and healthy controls.

The current meta-analysis has a number of limitations. One consideration is the potential effect of medications on social cognition deficits in ADHD. We were not able to explore the relationship between stimulant use and the observed impairment in social cognition due to lack of details and paucity of medication-naive samples. However, many of the studies included ADHD subjects that had been taken off their

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medications within the last 24–72 h, suggesting that impact of medications as a confounder of performance was relatively minimized. Another limitation was the lack of sufficient data to explore the effect of neurocognition other than IQ on between-group differences in social cognition tasks. Also, we were not able to explore differences in social cognition between ADHD and ASD in adults as only two of the studies included compared two patient groups in this age group. We were not able to include social cognitive domains other than emotion recognition and ToM in ADHD as no sufficient published data were available. Finally, our findings only reflect cross-sectional comparisons, as there were no available data to investigate development trajectories of these deficits in ADHD.

As a conclusion, ADHD is associated with social cognitive deficits with medium effect sizes, which are less severe than in ASD. Our findings also suggest that impairments in social cognition might improve with age in ADHD. Our findings indicate the need for longitudinal studies investigating the development of social cognition in ADHD.

Supplementary material

For supplementary material accompanying this paper visit http://dx.doi.org/10.1017/S0033291715002573

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Declaration of Interest

Over the last 2 years, C.P. has participated on advisory boards for Janssen-Cilag and Lundbeck. He has received honoraria for talks presented at educational meetings organized by Janssen-Cilag, Astra-Zeneca, Shire and Lundbeck. E.B. has no conflict of interests to be reported. Neither of the authors has financial or personal relationships, interests or affiliations relevant to the subject matter of this paper.

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